not have an anatomical basis; in fact, I seriously doubt that it is possible to recognize with any method of physical examination an amount of fluid less than 100 cc. In only one-seventh of the cases, therefore, would the amount of fluid present have been of diagnostic aid.

One is not always able to demonstrate the etiological factor of this disease. It is our impression that acute rheumatic fever, chorea and tonsil infection with the endocardial and myocardial complications account for the majority of our acute clinical pericarditis. Streptococcus viridans infection is closely associated with this group, whether as the primary infecting agent or not, it is difficult to say, but certainly it is a factor; eliminate this streptococcus and there is nothing left. Until proved otherwise let us regard this infection as the etiological one for this group. Our actual findings, however, would indicate that acute endocarditis was present 10 times with acute pericarditis; but on 32 other occasions no pericardial lesion was associated. This merely shows that endocarditis is the more common lesion of the two. On 6 occasions during the acute stage of rheumatic fever with arthritis and twice associated with acute chorea a pericarditis was found, and in all of these instances there was other cardiac involvement. Two acute infections of the throat in chronic heart and kidney disease completes the list of this etiological group, comprising thus two-fifths of the cases.

It was somewhat surprising to find that in 128 cases of lobar pneumonia there were only 10 associated with pericarditis, and in one-third of these the inflammation was purulent. It is of interest to remember that a considerable amount of suppurative pericarditis was found in the pneumonias of streptococcic origin; but this bacterial finding has been practically unknown in the lobar pneumonia of our community. The pneumococcus seemingly is not especially prone to develop a pericarditis (7 per cent), but when this condition does occur it is liable to become purulent. In all probability if the duration of pneumonia were longer more purulent exudates likely would be found in the pericardial cavity. There were 13 other cases in which some inflammatory focus was present in the lung, as foreign body with mediastinitis, empyema, abscess and gangrene, tuberculosis and cancer; about one-half of these were of streptococcic origin. Suppuration in the pleural sac is certainly a commonplace complication of pneumonia as compared with pericarditis in the same disease. One case of pericarditis was present in 50 autopsies on epidemic influenza.

Infection arising from the peritoneal cavity, particularly from the gall-bladder with liver or subdiaphragmatic involvement, accounted for 7 cases of pericarditis. In this instance the infecting organism is almost always a streptococcus pyogenes.

Recently, Libman has called attention to the association of acute pericarditis with coronary embolism and infarction of the heart.

I have confirmed this on one occasion during the past few weeks and there is one other case in our records. This observation would bear further watching, as it may be of considerable value in diagnosis.

It was not possible to get an accurate estimate of the bacteriology, as certain difficulties were present which would have to be considered before laying too much stress on the autopsy bacteriological findings. To put any reliance upon these findings it is necessary to have the cultures made within as short a time as possible after death, as post-mortem contamination in the thorax occurs very quickly. In our opinion, however, the Streptococcus viridans and pyogenes are the two common invaders while the pneumococcus ranks next. A word possibly would not be out of place regarding the fibrin deposited on the pericardial surfaces. It is often present in very small amounts. The surface over the right ventricle and at the base between the larger arterial trunks appears to be the most frequent situation for fibrin, especially when it is very sparingly distributed. In this connection it is of interest to remember that this is also the situation (anterior wall of right ventricle) favored by milk spots to which we previously referred. One has often to look carefully to see the fibrin on the surface of the right ventricle even where in life a well-marked friction sound had been heard. I have had the opportunity of following this point in several cases within the past year, and must admit being surprised to find so little fibrin in certain instances where very definite friction rubs had been present.

TABLE II.—CHRONIC PERICARDITIS.

Cases.	Sex.		Age.		General adhesions.	Local adhesions.
40	Males . .	31	Up to 10 . .	1	17	23
	Females .	9	10 to 20 . .	5		
			20 to 30 . .	0		
			30 to 40 . .	10		
			40 to 50 . .	5		
			50 to 60 . .	6		
			60 to 70 . .	2		
			70 to 80 . .	2		

The presence of fibrous adhesions in the pericardial sac indicates a chronic pericarditis. These adhesions may be localized as a few fine fibrous strands more often noted at the base of the heart, but also present anteriorly near the apex; or they may be diffusely present, forming the so-called general adhesive pericarditis. The localized fibrous adhesions in all probability in the majority of instances are little more than interesting pathological findings, as they are not only impossible to diagnose clinically, but there seems to be, further, little evidence to indicate that they actually produce any mechanical disturbance. Of course, a heavy isolated band in certain situations might produce considerable impairment in function leading to compensatory changes, as hypertrophy of the heart.

But these instances in our opinion are rather the exceptional findings. Table II shows that a little more than 4 per cent of the autopsies presented evidence of chronic pericarditis, and of this number a little less than half were of the general adhesive type. The male predominates, the relation being about the same as it is in the acute form of the disease. It is of interest to note that in one newborn child a diffusely adherent pericarditis was found. The infant presented some suggestive signs of congenital syphilis, although positive proof was never established. Over a half of the cases occurred under the age of forty, which is exactly opposite to what was found in acute pericarditis. The amount of fluid in the pericardial sac does not appear to have any relation to this form of pericarditis, as in the adherent form there is, of course, no fluid present, while in the localized type there is nothing in the adhesions themselves to call forth an effusion. It is my impression that one rarely sees chronic pericarditis with effusion, as this condition is either a persistent acute serofibrinous form or a tuberculous process. The latter is by no means rare.

It is a very difficult matter in many cases to identify the etiological factor responsible for chronic pericardial adhesions. This is but to be expected, as fibrous adhesions indicate an end-result, and it is always difficult to follow the course of a disease, especially pericarditis, a disease so often of an indefinite character. As a general principle, it would seem to us that the best explanation for the production of chronic adhesions is the organization of an acute fibrinous pericarditis and, therefore, the etiology would be the same for the chronic as for the acute form. Wells has suggested that a certain number of the adherent types of pericarditis are of a tuberculous nature, many possibly never existing in the state of a tuberculous pericarditis, but representing the fibrous tissue reaction induced by the tuberculous toxin developing in an adjacent active lesion such as the mediastinal lymph nodes. According to Wells the adhesive forms show nothing which would indicate tuberculosis either in the gross or in sections. We agree with this statement. It is possible that some obliterated pericardial sacs develop in the way suggested by Wells, but it is our impression that they form only a very small percentage of these cases. In but 3 of our 17 cases does it appear likely that tuberculosis was the etiological factor. At least it was the only disease present, the heart being perfectly normal. It should be added that there was nothing in these 3 cases in the pericardium itself to indicate morphologically a lesion characteristic of tuberculosis. According to our records this form of pericarditis is more often associated with endo- and myocarditis or what we may term the rheumatic-fever-cardiac group. This, in our opinion, is the etiological factor of first importance for any form of chronic pericardial adhesion but actual proof of this is not always forthcoming. That the other acute infections, as pneumonia, play

a part is but logical. There is nothing to indicate why general pericardial adhesions develop in some instances and local adhesions in others, although it would seem most likely that a massive acute inflammation would be more prone to develop general adhesions. On the other hand, however, it is quite possible that a recurrent infection could produce the identical result; while localized adhesions most likely would indicate single infections, probably not of an intensive character. As these are points which, even at the best, are debatable, one should not lay too much stress upon them.

Heart changes are not always associated with chronic pericarditis, and *vice versa*, pericarditis is not always present in heart disease; the endocardium and myocardium are undoubtedly more vulnerable than the pericardium. This fact is brought out in our records which show no pericardial involvement in 43 autopsies in which the cause of death was due to heart disease. In 11 of the chronic adhesive pericarditis cases there was evidence of chronic endo- or myocarditis, thus leaving 3 cases of this type in which the etiology was most indefinite, as tuberculosis was not present. The relation of kidney and arterial disease to chronic pericardial changes is approximately the same as that of the heart, in that these three vital structures are so commonly associated in disease. The actual size of the heart does not appear to depend entirely upon mechanical difficulties thrust upon it by an adherent pericardium, as there were several instances with little or no cardiac hypertrophy. Enlargement of the heart in this condition is seemingly related more to the myo- or endocardial changes, to nephritis with hypertension or to extensive adhesions to the adjacent structures outside of the pericardial sac. There were no typical examples of Pick's disease in our series.

TABLE III.—TUBERCULOUS PERICARDITIS.

Cases.	Sex.		Age.	
10	Males	0	Up to 10	0
	Females	1	10 to 20	2
			20 to 30	3
			30 to 40	2
			40 to 50	2
			50 to 60	1

In this table one sees again the predominance of the males in pericarditis and it further indicates an earlier age level, as three-fourths of the number were met with before the age of forty. Tuberculous pericarditis usually produces a very large sac, containing a great deal of fibrin and often a large amount of fluid. This fluid may be blood-tinged, but more often it is clear and straw colored. The largest pericardial sacs, according to our records, have been the tuberculous ones. One rarely sees at autopsy the development alone of miliary tubercles on the pericardial surfaces in the same manner as one observes them in the pleura. Certainly, there is probably

a stage in the development of tuberculous pericarditis when it is likely that only tubercles are present; but the production of fibrin is soon so extensive that, at first glance, the characteristic appearance is that of an acute fibrinous pericarditis. At autopsy this fibrinous character is always evident, and it is by no means improbable that a certain number of tuberculous pericarditis cases are labelled acute fibrinous pericarditis. The tubercles are readily enough seen when the fibrinous exudate is scraped off so as to expose the deeper layers, and once in a while it is necessary, when the tubercles are indistinct, to wait for a microscopic section in order to be quite sure of the diagnosis. The term bread-and-butter heart may apply equally well to both the acute fibrinous and the tuberculous forms of this disease. At times one sees in the deeper portions of the exudate the development of a moderate amount of caseation, although this feature has not been prominent in the materials which we have studied.

The question has been brought up as to whether some of the diffuse adherent pericardial sacs represent healed forms of tuberculosis. Although this is a possibility it is my impression that such a fortunate sequence of events rarely occurs. The healed tuberculous lesions of the pericardium which present unmistakable signs of this disease are extremely unusual if one is to consider calcification as an evidence of healing. Wells observed calcification on 4 occasions, but he is inclined to look upon this deposit as being secondary to a suppurative condition in the sac. We did not see any example of calcification in our series, but agree with Wells that this change and probably also what has been described as calculi in the pericardial sac represent the end-result of a purulent pericarditis. Calcification following chronic empyemas secondary to lobar pneumonia with tuberculosis ruled out at autopsy, in which a cast of the chest wall had formed, have occurred several times in our records. So by analogy it would appear that the same process is possible in the pericardial sac. We mentioned before Weil's conception of the relation of tuberculosis to certain of the adhesive forms of chronic pericarditis. He admits that it is a difficult thing to prove, and we follow him in this matter.

A similarity of origin is quite possible between adherent pericarditis and chronic adhesive pleuritis. It is the general conception that tuberculosis is the most common productive factor in this latter condition, although by no means the only factor. Anatomically these adhesions represent nothing but fibrous tissue, and only rarely is one able to demonstrate in them histological evidences characteristic of tuberculosis. There is, therefore, some ground for considering chronic adhesive pericarditis as having a tuberculous etiology, but as we have seen this condition associated more often with the rheumatic group it is our impression that tuberculosis accounts for but a minimum number of these cases. It would be interesting to observe

the final result in the cases of tuberculous pericarditis which have been apparently cured by drainage of the sac. My experience with this disease has led me to regard it as a very severe form of local tuberculosis possibly as malignant as its kindred infection of the meninges.

The relation of pericarditis to tuberculosis elsewhere in the body is of considerable interest. It is in fact always a secondary infection. There was only one case in which we were unable to find the primary focus of tuberculosis, but this by no means indicates that the pericarditis was the primary disease; it merely shows that the original focus was probably very small, likely a hidden lymph node lying so close to the pericardium that it could not be clearly identified. A point of considerable interest is that the development of a tuberculous pericarditis seems to have little relation to the extent of the tuberculous process elsewhere. In other words this disease in the pericardium is often purely a local entity in the same way as tuberculosis of the peritoneum or meninges. Remembering how prone endothelial lined cavities are to involvement in a process of general miliary tuberculosis, it was of interest to record but 3 cases where a pericarditis was present in this general infection. The pleura, peritoneum, meninges and solid organs may be studded with tubercles and yet the pericardium escape; while, on the other hand, a marked pericarditis may be present with only the mediastinal lymph nodes presenting the primary lesion. It appears to us that tuberculous pericarditis has a more intimate etiological relationship to tuberculosis in the mediastinal lymph nodes than to this disease in any other situation, and that the infection is lymph-borne or possibly by direct extension. This view is in agreement with what has been previously expressed by many writers on this subject. A blood-stream infection in general miliary tuberculosis does occur, but this we regard as the rare method of infection of the pericardial cavities. In only 5 cases out of a total of 37 dying of pulmonary tuberculosis was there any evidence of a tuberculous pericarditis. This would seem to indicate that the pericardium very often escapes even in fatal forms of tuberculosis of the lung. As latent or healed tuberculosis of the lung is such a common finding, its relation, therefore, to the pericardium must be practically *nil*. As was just stated, in 3 instances of general miliary tuberculosis there was a tuberculous pericarditis, but in 20 others, comprising miliary tuberculosis of the peritoneum, meninges, pleura and bowel, the pericardium was negative. Both the peritoneum and the meninges are more often involved while the pleura is rarely missed in a tuberculous death.

Diagnosis. *Acute Pericarditis.* Pericarditis is often not diagnosed, as the clinical record of any service, where autopsies are made with moderate regularity, will show. It is no doubt a difficult diagnosis to make in many cases. Further, some of the very recent

inflammations of the sac found at autopsy are probably of relatively few hours' duration, hence they could be very easily missed. It is also possible and probable that some of these infections do not give any physical sign. At the same time, however, we are quite certain that many acute pericardial cases which are unrecognized, at some stage of their course do present the classical signs, and if one is on the watch for this disease, positive results will be more often forthcoming. We have nothing new to add to the method of diagnosis; the symptoms and signs have been described in detail in text-books and in numerous articles.

In the diagnosis of acute pericarditis the friction rub is by far the most important sign. We do not need to go into its character or its situation, but suffice it to say that the to-and-fro grating sound, heard usually outside of the left sternal border below the third rib, is pathognomonic of a fibrinous pericarditis. The rub may be localized to an area not larger than the end of the stethoscope or it may be well heard over the whole of the precordium. Cessation of breathing and slight pressure on the end of the stethoscope are aids in bringing out this murmur. Its demonstration is worth all of the other symptoms and signs combined. The fleeting character of the friction sound should be kept in mind, as it may be present when the intern makes his night rounds and yet be absent when he attempts to demonstrate it to his chief in the morning. This is the reason why careful and frequent observations are essential if we are going to recognize the majority of the acute fibrinous pericarditis cases. I am not sure that the development of fluid is the only reason to explain the disappearance of the friction rub. It is possible that where very little fibrin has formed, absorption may readily occur. A very loud rub can be produced by an unusually small amount of fibrin, so small, in fact, that it may be readily missed by a careless gross inspection of the heart at autopsy. The next sign of value appears to be the size of the cardiac dulness. The increased dulness is due to the presence of fibrin or fluid, or both. One must keep in mind, however, that an increase of fluid is by no means a regular finding in acute pericarditis. Half of our cases had less than 100 c c, and I doubt very much that this amount could be appreciated by physical examination. Possibly as small an amount of fluid as this might be recognized by a roentgen-ray plate or fluoroscopy. Where larger amounts of fluid are present the well-known pear-shaped cardiac dulness can be readily demonstrated by percussion, but we should always confirm this with roentgen-ray examination. This form of pericarditis with its sudden onset and rapid accumulation of fluid, as shown by the increase in the cardiac dulness, can be readily followed in its development. Associated often with this rapid distention of the pericardial sac, one may observe cyanosis and dyspnea accompanied by pain.

The change in the intensity of the cardiac sounds from the very

distinct to the faint or muffled character, as the fluid and fibrin accumulates can, at times, be observed, and when present is of great value.

The presence of the precordial pain, associated with a febrile condition and an increased pulse-rate, is seen in about a third of the cases. McKenzie believes that all pain accompanying acute pericarditis is a manifestation of myocardial involvement, as the lesions of the pericardium itself are painless. This is a very interesting point, and may be the explanation, although McKenzie in his typical way does not force us to accept his conclusion. Might it not also be possible to explain the pain in some cases of pericarditis as being due to an associated involvement at the base of the aorta? Klotz has called our attention to the fact that an aortitis is not an infrequent sequela of an acute rheumatic fever. As it is following this disease that most of our acute clinical pericarditis develops, it would seem reasonable to believe that aortitis, which is very often painful, may share with the myocardium as being the source of this important symptom. There is one other explanation to be considered: the sudden stretching of any moderately rigid sac is liable to cause pain, and, therefore, in those cases of acute pericarditis associated with the rapid accumulation of fluid the presence of pain may be due to the stretching of the pericardium. This, however, does not appear to apply if enlargement of the sac has not been brought about rapidly, for we have seen enormous pericardial sacs in tuberculous pericarditis which clinically were quite devoid of any suggestion of pain. These accumulations, however, are always of a more gradual development.

There are many other minor signs described, but it is unnecessary to repeat them here, as Robey has recently written on this matter in detail, to which work the reader is referred.

Chronic Pericarditis. The diagnosis of chronic pericarditis is a very difficult one to make, much more so than the acute form. It may, in fact, be impossible to recognize this condition. We can see no clinical method which could possibly help us to diagnose local isolated fibrous adhesions. This form of pericarditis is purely an autopsy finding, but where the adhesions are general, or massively developed at one point, they may produce in a certain number of instances signs by which they can be recognized. In view of the fact that some cases of general adhesive pericarditis are found at autopsy without evidence of their having produced any change in the gross appearance or function of the heart, it can be readily seen that there are difficulties ahead when one attempts to speak on the diagnosis of chronic adhesive pericarditis. There are no pathognomonic signs or symptoms of this condition, but there are some suggestive points which when present, are of value, but their absence is not of much significance in ruling out adhesive pericarditis.

The retraction of the lower ribs, posteriorly on the left side, with

cardiac systole (Broadbent's sign) is probably of most value in diagnosis although it must be kept in mind that this sign has been found in other conditions than adhesive pericarditis. The retraction of the lower end of the sternum and the pulling in of the costal margins in inspiration, as pointed out by Hoover, are at times noted. There are a number of other points that have been suggested, and possibly on certain occasions they are of value. The diastolic shock at the apex and the diastolic collapse of the distended veins in the neck have been given as signs of clinical worth, but I doubt if anyone would like to rely too much on such evidence. It is our impression that fluoroscopic examination of the diaphragmatic movements in a certain percentage of cases may offer some suggestive points of value. It might also be of importance in roentgen-ray examinations to lay more stress on the appearance of the mediastinal tissues which usually show some alteration when the pericardial sac is involved. No reliance is placed upon the quality of the heart sounds, but cardiac derangement out of keeping with the apparent valvular lesions has been advanced as possible evidence of adhesive pericarditis.

There is a point which I believe may be of great value in making this diagnosis, but it is neither a sign nor a symptom. To be able to prove without any question of doubt that the individual had a previous attack of acute pericarditis is of extreme value, and in our opinion may be the only factor present which would lead one to the diagnosis of adhesive pericarditis. We have but recently seen an excellent example of this very condition. Five years ago, in the medical ward, I observed a very extensive acute fibrinous pericarditis following an acute rheumatic fever and associated with an endocarditis and in all probability a myocarditis. The patient recovered and graduated as a male nurse a few years afterward. He was in good condition and worked regularly at his occupation. He had some suggestive signs of a mitral stenosis with regurgitation; the heart was hypertrophied, but the action was regular. In April of this year, five years after the original infection, he developed tonsillitis with an acute pleurisy and atrial fibrillation was recognized. It was not present a short time previous to this acute attack of infection. He made a perfect recovery from his acute conditions and after a month's rest had his tonsils removed under local anesthesia. He died suddenly a month later. At autopsy a general adhesive pericarditis with hypertrophy and dilatation of the heart and a slight thickening with but little stenosis of the mitral valve were found. There was nothing whatever in this case, in the way of physical sign, pointing to an adhesive pericarditis except that we known positively that some years previous he had an acute fibrinous pericarditis, and on this ground alone an adhesive pericarditis had been suggested along with the myo- and endocardial changes.

Tuberculous Pericarditis. The form of tuberculous pericarditis which we especially refer to is a serofibrinous exudate implanted

upon a tuberculous base. Some of the largest pericardial sacs and some of the best examples of the bread-and-butter heart are of this type. Clinically, tuberculous pericarditis very often proceeds as an individual process which early has the appearance of an acute pericarditis, but which later passes into the subacute or chronic form. In our experience they have all been fatal and not necessarily associated with marked or extensive tuberculosis elsewhere. In a chronic pericarditis with effusion one should always think of tuberculosis, and here, of course, it must clearly be understood that a hydropericardium is not to be mistaken for this condition. The diagnosis of tuberculous pericarditis is not always easy, and at times is made only at autopsy. The chief point in diagnosis is the chronicity of the process in a lesion which presents the physical signs of an acute serofibrinous pericarditis.

In the early stages the signs are like those of acute fibrinous pericarditis. The friction rub is usually well heard over a considerable portion of the precordium, and it often persists for a considerable period of time. On two occasions I have been able to hear the rub for almost a month. There is rarely any pain in this condition, although it may be present. A diffuse, persistent friction rub over the heart without the slightest evidence of pain, as is seen in many of those cases, rather supports McKenzie's statement that the pericardial sac is devoid of pain. As fluid is nearly always present, and at times in very large amounts, the cardiac dulness consequently is much increased. The presence of dulness behind at the left base is noted in this condition with some regularity. It is not diagnostic of the tuberculous pericarditis, as it may also be present in any type of sac containing a large amount of fluid. The change from day to day in the character of the heart sounds is of considerable value, and I have been able to follow this sign on several occasions from clear, loud cardiac sounds to those which became distinctly muffled. The accumulation of fluid and fibrin is responsible for this change in the heart sounds. The febrile reaction is of a tuberculous type. The pulse-rate is rapid, and as the disease advances the temperature elevations become greater and the rapidity of the pulse increases. The course is progressively downward.

Conclusions. 1. Ten per cent of our autopsies showed pericarditis; 5 per cent were acute, 4 per cent were chronic and 1 per cent were tuberculous.

2. The principal etiological factor, in the acute and chronic forms of pericarditis, seems to be associated with the disease acute rheumatic fever.

3. Acute pericarditis was present in 7 per cent of the acute lobar pneumonias. The majority of these appeared probably shortly before death. The streptococcus in thoracic infection is more prone to produce suppuration in the pericardial sac than the pneumococcus.

4. In 54 cases of death due to tuberculosis there was no evidence of tuberculous pericarditis.

5. Tuberculous pericarditis is usually of the scrofibrinous variety; it may be present in the absence of other clinical signs of tuberculosis; pathologically there is always a primary focus, usually in the lymph nodes of the thorax or in lung. The pericardium is often missed in a general miliary tuberculosis.

6. Tuberculous pericarditis should always be considered when the signs of acute pericarditis persist for an unusually long time, and especially when there is considerable cardiac enlargement and a progressively downward clinical course.

7. The presence of a friction rub is the most valuable diagnostic sign of acute pericarditis.

8. Chronic adhesive pericarditis can be diagnosed only in a small percentage of cases, as there are no distinctive signs. It is of real value to know of a previous undoubted attack of acute fibrinous pericarditis when considering the possible presence of a general adherent pericarditis.

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BALANTIDIUM COLI AND PERNICIOUS ANEMIA: REPORT OF FOUR CASES.

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Balantidium coli is a flagellate parasite inhabiting the colon, common in animals, especially the pig, but rare in man. The work of Strong, Bowman, Bel and Couret, and Manlove, shows that the parasite is capable of invading all layers of the intestinal wall. It has been found in the bloodvessels of the intestine and in other organs. With the accompanying bacteria superficial and deep ulcers are formed which cannot be distinguished from ulcers due to ameba until the microscope differentiates the parasite. The parasite also possesses the power of encysting.

The clinical symptoms of invasion by *Balantidium coli* bear a great similarity to those produced by ameba. Both present an acute and chronic form. In the acute form there is severe bloody dysen-

tery, with great loss of fluid and marked toxicity. Perforation may occur. Death follows usually in from a few days to one or two weeks. The chronic form often exists for years with alternating constipation and diarrhea. As with many other parasites the acute form seems to develop chiefly in warm countries and the chronic form in colder regions.[10] The symptoms and findings of pernicious anemia seem to accompany the chronic form of the infection.

The prognosis in the acute type is very bad, even if the case is seen early. In the chronic type it seems to be bad only when the syndrome of pernicious anemia is added. Our patients have died from the pernicious anemia rather than directly from the dysentery.

Schwartz finds a hemolytic agent in *Ascaris lumbricoides* and *Trichuris trichiura*, which he believes accounts for the anemia in these infections. Under certain conditions, supposedly including a decomposition of the segments of the worm, *Dibothriocephalus latus* produces a picture of pernicious anemia. If the worm is expelled the blood picture returns to normal.

Glaessner has found a hemolytic agent in *Balantidium coli*. It seems probable that the constant subjection of the blood-forming organs to the poison is the cause of the pernicious anemia syndrome in the chronic type of case. The removal of the *Balantidium coli* and its hemolytic agent is a far different problem than the removal of the Dibothriocephalus latus. Since *Balantidium coli* penetrates the intestinal wall and its bloodvessels and other organs in the body and also encysts we cannot be sure when the organism is eradicated from the stools that it is not harbored somewhere in the tissues of the body with its hemolytic agent still active. Thus if the pernicious anemia complex persists after treatment we cannot be sure whether the toxin of *Balantidium coli* is still active or whether once the complex is started it becomes an entity.

The treatment resolves itself into ridding the intestinal lumen and the body tissues of the organism. The literature indicates that most of the drugs used against intestinal parasites are without effect on *Balantidium coli*. In our experience in the Clinic with the flagellate *Lamblia intestinalis* we found that the only drug that gave any satisfaction was put into the blood in sufficient quantity to act as a direct poison when ingested by the parasite; in a large number of cases arsphenamin proved of great value. In our latest case of infection with *Balantidium coli* we employed arsphenamin with apparently very good effect. An enema with vinegar and tannic acid also was given every day or every second day with the idea of producing an acid medium in the colon and of giving a direct parasiticide. On account of encystment the treatment must be carried out a long time, just how long I do not know, since the length of time the organism may remain encysted and become active again has not been determined. That the living organism is very resistant is shown by Klein, who found actively motile *Parame-*

cium coli in sewage which had been bottled for three weeks. In the case treated by five intravenous injections of arsphenamin at weekly intervals, nine stool tests were negative for the parasite, the diarrhea was entirely checked, and the patient felt very much better; yet the neurological symptoms of pernicious anemia still persisted. Jennings reports one case with the pernicious anemia syndrome which was apparently cured after two relapses.

It is interesting in connection with the family histories of three of our patients that death of one or more parents, brothers or sisters, persons in the same household living under similar conditions and eating the same food, occurred from pernicious anemia or anemia. This may explain the family tendency to pernicious anemia in many cases.

Case A52257, Mr. W. T. L.; aged forty-two years, a farmer for the past five years, came for examination April 26, 1911. The patient had had a spell of diarrhea for two months when working in a butter and cheese factory twelve years before. The diarrhea had recurred off and on until five years before, when it became more constant and severe; blood was passed at intervals. During the past one and one-half years the patient had lost 10 to 15 pounds in weight and had become weak and dyspneic on exertion. One year before sore-mouth developed, at times severe enough to make eating difficult. During the past one and one-half months numbness and tingling had been noticed in the hands and feet.

Examination. The examination of pus from around the teeth revealed many motile ameba and two organisms from the Flagellata, one evidently Trichomonas, the other narrower and much longer than Trichomonas. The total gastric acidity was 8; free hydrochloric acid 0. In the examination of the stool large numbers of *Balantidium coli* but no ameba were found. The blood picture is shown in Table I.

Treatment. Blaud's pills, Fowler's solution, and rectal injections of vinegar and tannic acid were given. For six months there were marked improvement and gain in weight with a great reduction in the number of *Balantidium coli* in the stools. Reëxamination after nine months showed no *Balantidium coli* in one stool test, but the clinical symptoms and blood picture of pernicious anemia were still present.

Case A185278, Mr. G. A., aged thirty years, a farmer who had never been outside of Illinois, Minnesota, and Iowa came for examination February 9, 1917. The patient's father had died of pernicious anemia one year before. Nine years before and two years before the patient had had short attacks of diarrhea. Five weeks before he had had "grippe" and one week later he had had a sudden attack of pain at the right costal margin running up into the chest

BLOOD FINDINGS IN FOUR PATIENTS HAVING BALANTIDIUM COLI

	A52257		A185278			A215036		A21480			
	4/26/11	1/2/12	2/10/17	3/12/17	9/28/17	11/27/17	12/21/17	5/5/20	5/10/20	8/10/20	9/16/20
Hemoglobin (%)	35	43	35	70	40	44	48	25	30	58	55
Erythrocytes (millions)	1.0	1.07	2.1	3.14	1.60	1.0	2.70	1.97	2.14	2.80	2.84
Color index	1.1	1.0+	0.8+	1.1+	1.2+	1.3+	0.8+	0.0+	0.0+	1.0+	0.9+
Leukocytes	10,400	9,700	11,800	0,400	3,600	4,600	5,000	0.800	6,000	4,400	4,000
Cells counted	300	300	300	300	300	300	300	200	200	200	200
Polynuclear neutrophils	58.6	00.7	60.3	64.3	54.7	55.3	44.3	07.0	30.0	48.5	30.0
Small lymphocytes	32.4	20.0	20.0	20.3	37.7	33.7	45.3	20.0	53.0	41.5	58.0
Large lymphocytes	4.0	1.7	6.3	4.7	4.3	1.7	6.3	4.0	7.0	7.0	3.0
Eosinophils	5.0	1.7	1.3	1.7	2.7	8.7	3.7	....	0.5	2.0	2.0
Basophils	....	1.0	.....	....	0.3	0.7	0.3	....	0.5	1.0	1.0
Neutrophilic myelocytes	....	....	....	....	0.3						
Normoblasts	4.0	1.0	1.0	....	8.0	....	....	....	1.0		
Megaloblasts	8.0										
Poikilocytosis	Marked	Marked	Moderate	Moderate	Marked	Moderate	Marked	Moderate	Moderate	Moderate	Moderate
Granular degeneration of erythrocytes	Slight	....	Moderate	Slight	Marked	Moderate	Moderate	Moderate	Slight	Slight	Slight
Polychromatophilia	Slight	....	Moderate	....	Slight	....	....	....	....	....	Slight

and right shoulder and difficult breathing. Diarrhea had been constant since the attack of grippe, with from four to five loose stools in the early morning and at night.

Examination. The patient was pale and sallow. He had pyorrhea and much bridge-work in his mouth. The systolic blood-pressure was 116, the diastolic 54. The urine was negative for bile and urobilinogen, but urobilin was present. Roentgenograms of the chest and teeth were negative. The blood picture is shown in Table I. The duodenal contents showed a brown trace of bilirubin, urobilinogen 1000 units, and urobilin 1400 units, indicating increased blood destruction. The examination of the central nervous system was negative. Six stool tests showed *Balantidium coli* but no other parasites.

Treatment. Thymol and Blaud's pills by mouth and enemas of kerosene were given; in three weeks this man eliminated almost all of the *Balantidium coli* and raised the hemoglobin from 30 per cent. to 70 per cent. The patient went home feeling much improved.

Examination six months later showed the *Balantidium coli* still in the stools and the clinical picture and blood findings of pernicious anemia. Examination of the central nervous system showed the cord changes usually found in pernicious anemia. The patient was again treated with thymol, salol, Blaud's pills, and Fowler's solution, but he left for home after three weeks, unimproved. He died the following summer.

Case A215036, Mr. G. H. B., aged fifty-two years, a farmer, was examined November 26, 1917. The patient's father, brother, and sister died of similar trouble, "thin blood." For twenty years past the patient had had spells of pain in the upper abdomen lasting for from one to two hours, with vomiting. He had had malaria at the age of fifteen. During the past year diarrhea had been persistent, five to six stools daily. He had lost about 60 pounds in weight; he had lost strength progressively and in the past three months numbness over the entire body with tingling in the extremities had come on. His appetite was poor; he had not had a sore tongue. His memory had failed for two months and recently he had been irritable, and had had crying spells.

Examination. The blood picture may be seen in Table I. The systolic blood-pressure was 150, the diastolic 100; the pulse 84. Examination of the urine was negative. The total gastric acidity was 10, free hydrochloric acid 0. Roentgenograms of the stomach were negative. Two blood Wassermann tests were negative. Proctoscopic examination showed very slight proctitis for 14 inches. The stool test revealed *Balantidium coli* but no other parasite. Examination of the central nervous system showed subacute combined selerosis.

Treatment. The patient was treated for two months with thymol, chloroform, Blaud's pills, and hydrochlorie acid without improvement. The *Balantidium coli* persisted in the stools and the blood picture remained the same. The patient died two months after leaving the Clinic.

Case A21480, Mr. F. L. S., aged fifty-seven years, a farmer, was examined in the Clinic May 4, 1920. One sister had died of anemia at forty-eight. The patient had come to the Clinic eleven years before on account of weakness and loss of strength with spells of diarrhea, at times severe. One stool test had showed occult blood. The protoscopic examination had been negative. During the following years the patient had had spells of diarrhea every ten days, lasting four or five days, with four or five watery stools a day, usually in the morning. Between attacks the stools were normal. Eight months before examination the patient had returned from the state fair with diarrhea and abdominal cramps. He noticed numbness in the extremities, his feet felt "asleep, or as if varnished," and had some weakness of the anal and vesical sphincters.

Examination. The patient's systolic blood-pressure was 116, diastolic blood-pressure 78, pulse 84. The test of the urine and the Wassermann test on the blood were negative. The total gastric acidity was 46, the free hydrochloric acid 0. Foci of infection were not found in the teeth or tonsils.

After tests on the stool and blood were made in the Clinic the patient went away for two days to visit relatives; he was brought back on a cot, with hemoglobin of 25 per cent.

Treatment. The patient was treated with distilled chenopodium and he improved in every way. The number of Balantidia was greatly reduced. The patient was given enemas of vinegar and tannic acid every second day and arsphenamin intravenously once a week for five weeks. After the last treatment nine stool tests, August 5, 6, 12, 13, 19 and 20 and September 16, 17 and 18, failed to show any Balantidia. The clinical symptoms of pernicious anemia are still present and the last blood test, September 16, showed 55 per cent. hemoglobin.

The examination of the central nervous system on three occasions is interesting in showing progressive involvement. May 11, when the hemoglobin was 30 per cent, the neurologist reported slight subacute combined sclerosis, hardly as extensive as in the average case of pernicious anemia of the same severity. July 14 the neurologist reported evidence to be insufficient for a diagnosis of a central nervous system lesion, but findings of combined sclerosis. August 21 the report was subacute combined sclerosis of the pernicious anemia type. This last report was made by a consultant who had not examined the patient previously.

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SOME EXPERIENCES WITH THE MELTZER-LYON METHOD OF DRAINING THE BILIARY SYSTEM.

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Investigations have been conducted during the past year with clinical cases, operation observations and animal experimentation pertaining to the Meltzer-Lyon method of aspirating duodenal contents after the installation of a solution of magnesium sulphate. This was done for the purpose of proving, if possible, the premises of Meltzer and Lyon as to the accuracy of published statements on the physiological phenomena of gall-bladder function and of this method of treating biliary conditions. We feel that experimental studies on the physiology of influencing motor phenomena of the gastro-intestinal canal or the associated organs in animals cannot be applied, except in a general way, to the physiology of the digestive tract in man. It follows then that clinical and operative experience, when these are obtainable and carefully controlled and deducted from, are more accurate and worth the while, and therefore none of our animal experimentation is presented.

S. J. Meltzer[1] published an article in which he drew attention to the supposed function of the sphincter at the papilla of Vater and

[1] Am. Jour. Med. Sc., 1917, cliii, 469.